TUTORIAL 4: ARTERIAL BLOOD GASES (ABG’s) and PULMONARY EMPHYSEMA

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READING: Porth, Chapter 32 (acid-base balance) and pp. 716-722 (COPD)

Also helpful:

References:

OBJECTIVES:

At the end of this tutorial, students should be able to:
1. describe normal arterial blood gas parameters.
2. recognise respiratory and metabolic acidosis and alkalosis, describe the pathophysiology that leads to these derangements, and explain the principles of treatment for these disorders.
3. explain the mechanism of respiratory and metabolic compensation of acid-base imbalances.
4. describe the aetiology and pathophysiology of pulmonary emphysema, and use this information to explain the manifestations, diagnosis and treatment of pulmonary emphysema.
5. describe how chronic bronchitis and pulmonary emphysema differ in presentation and explain those differences.

INTRODUCTION:

Arterial blood gases can be used to detect respiratory and metabolic abnormalities and treat acid-base imbalances. The values examined are PaO₂ (called PO₂ in your textbook), PaCO₂ (called PCO₂ in your textbook), pH, and HCO₃⁻. Remember that the point of pulmonary ventilation is to breathe oxygen (O₂) in, and to excrete carbon dioxide (CO₂).
**PaO₂ (arterial oxygen concentration)**
The normal value is between 80-100 mmHg. A lower value indicates hypoxaemia.

**SO₂ (arterial percentage oxygen saturation)**
This term refers to the percentage of oxygen binding sites on the Haemoglobin molecule that are loaded with oxygen. The affinity of the sites for oxygen varies with temperature and acidity, which is important for oxygen loading and unloading. The normal range is 95-100%.

**pH (a measure of hydrogen ion concentration)**
The normal range is 7.35 - 7.45. If the value is lower than 7.35, an acidosis is present. If the value is greater than 7.45, an alkalosis is present. A pH within the normal range may be due to compensation of an underlying disorder, so all values should be considered together.

**PaCO₂ (arterial carbon dioxide concentration)**
Remember that breathing out is how our bodies get rid of carbon dioxide, thus the PaCO₂ is controlled by the rate and depth of respiration. The normal range is 35 - 45 mmHg. A higher value causes an acidosis, whilst a lower value causes an alkalosis. This is because carbon dioxide combines with water to make carbonic acid. So, the slower and shallower your breathe, the more CO₂ you will retain, and the more acid your serum pH will be. On the other hand, if you hyperventilate (breathe fast and deep), you will blow off your CO₂ and your serum pH will rise (become more alkaline).

**HCO₃⁻ (Bicarbonate)**
Bicarbonate levels are controlled by the kidneys. The normal arterial range is 21 - 28 mEq/L. An elevated level causes an alkalosis, while a decreased level causes an acidosis. In times past people treated indigestion with a spoon of bicarbonate, because it is a base and thus neutralises acid, so you can see that the more bicarbonate we have in the blood, the more alkaline or basic conditions will be.

**There are a few general rules that can be used to identify abnormalities:**

1. If the pH is dropping and the PaCO₂ is rising or vice versa, a respiratory disorder is present:
   - ie. pH 7.3, PaCO₂ 50 mmHg = respiratory acidosis;
   - pH 7.5, PaCO₂ 25 mmHg = respiratory alkalosis.

2. If the pH and HCO₃⁻ are both rising or both falling, a metabolic disorder is present:
   - ie. pH 7.25, HCO₃⁻ 19 mEq/L = metabolic acidosis.

3. If the PaCO₂ and HCO₃⁻ are both rising or falling, the body is compensating for an imbalance:
ie. pH 7.3, PaCO\textsubscript{2} 20 mmHg, HCO\textsubscript{3}- 19 mEq/L (pH and bicarbonate levels are both low indicating a primary metabolic acidosis. The PCO\textsubscript{2} is decreased because the body is attempting to compensate for the acidosis).

4. If the pH is normal but the PaCO\textsubscript{2} and HCO\textsubscript{3} are abnormal, a fully compensated disorder is present. If the pH lies towards the acid end of the normal range, the primary disorder is an acidosis; if it lies towards the alkaline end of the range the primary disorder is an alkalosis:

   ie. pH 7.42, PaCO\textsubscript{2} 55 mmHg, HCO\textsubscript{3}- 34 mEq/L (metabolic alkalosis compensated by increased CO\textsubscript{2} retention).

5. If the PaCO\textsubscript{2} and HCO\textsubscript{3} are changing in opposite directions, a mixed imbalance is present (ie. in a cardiac arrest you will see both a metabolic and respiratory acidosis).

An easy way of determining acid-base balance from an ABG is to put next to each parameter whether it is increased or decreased and whether that makes conditions more acidic or alkaline. Match up the parameters that are both becoming acidic or both becoming alkaline. If the PaCO\textsubscript{2} is the cause then the condition is respiratory; if the bicarbonate is the cause then the condition is metabolic.

Eg.  pH 7.25 (this pH is acid as it is below the normal level)
     PaCO\textsubscript{2} 42 mmHg (this is within the normal range)
     HCO\textsubscript{3}- 19 mEq/L (this is acid as the bicarbonate which is an alkaline substance is lower than normal)

Thus, the bicarbonate level is abnormal, and both it and the pH are acidic, therefore the person has a metabolic acidosis.

There is a quiz on arterial blood gases in your online unit. Click on the unit link in MySCU, then click on the button ‘Assignments’, and click on the quiz button. You can do this quiz multiple times and it will give you new questions, and feedback on questions you get wrong, to help you learn this skill.

REVIEW QUESTIONS

1. Briefly describe the principle of homeostasis, using PaCO\textsubscript{2} levels and pH to illustrate how homeostasis works (in your answer you will need to discuss the receptors, control centre and effectors and how they control PaCO\textsubscript{2} levels and thus pH).
2. What is the purpose of a buffer system?

3. Hydrogen ions can trade places with which intracellular cation to normalise acid-base balance? What happens to serum levels of this cation if the blood pH falls below its normal range?

4. How do serum proteins act as a buffer for hydrogen ions and which cation can be displaced from the serum proteins when conditions are acidotic?

5. How is oxygen transported in the blood?

6. How do the kidneys help to regulate pH?
PART A: Interpret the following ABGs

**pH 7.37**
PaCO₂ 48 mmHg
HCO₃ 29 mEq/L
PaO₂ 70 mmHg
SaO₂ 93%
**Acid - base state:**
**Compensation:**
**Oxygenation:**
Possible problem?

**pH 7.52**
PaCO₂ 30 mmHg
HCO₃ 24 mEq/L
PaO₂ 90 mmHg
SaO₂ 98%
**Acid - base state:**
**Compensation:**
**Oxygenation:**
Possible problem?

**pH 7.48**
PaCO₂ 33 mmHg
HCO₃ 25 mEq/L
PaO₂ 68 mmHg
SaO₂ 95%
**Acid - base state:**
**Compensation:**
**Oxygenation:**
Possible problem?

**pH 7.38**
PaCO₂ 38 mmHg
HCO₃ 24 mEq/L
PaO₂ 180 mmHg
SaO₂ 100%
**Acid - base state:**
**Compensation:**
**Oxygenation:**
Possible problem?

**pH 7.30**
PaCO₂ 25 mmHg
HCO₃ 12 mEq/L
PaO₂ 85 mmHg
SaO₂ 94%
**Acid - base state:**
**Compensation:**
**Oxygenation:**
Possible problem?

**pH 7.49**
PaCO₂ 50 mmHg
HCO₃ 35 mEq/L
PaO₂ 82 mmHg
SaO₂ 96%
**Acid - base state:**
**Compensation:**
**Oxygenation:**
Possible problem?
pH 7.23
PaCO₂ 75 mmHg
HCO₃ 26 mEq/L
PaO₂ 60 mmHg
SaO₂ 82%

Acid - base state:  
Compensation:  
Oxygenation:  
Possible problem?

pH 7.22
PaCO₂ 57 mmHg
HCO₃ 19 mEq/L
PaO₂ 49 mmHg
SaO₂ 76%

Acid - base state:  
Compensation:  
Oxygenation:  
Possible problem?

PART B: CLINICAL PROBLEM

Pulmonary emphysema

Initial presentation

Mick, aged 60, presents to the hospital with acute respiratory distress. He is a current smoker, who has smoked for approximately 40 pack years (eg. a pack a day for 40 years, or 2 packs a day for 20 years).

Manifestations

- Thin, barrel-chested
- Heart rate 120 bpm
- Temperature 37.8°C
- Tachypnoea, dyspnoea
- Restless
- BP 140/80
- Sitting on the side of the bed, leaning over a bed table
- Cyanosis
- Pursed lip breathing

Past history

Mick has lost 5 kg in the last few months, and he has had a morning cough and shortness of breath throughout the day, but particularly on exertion. The patient has a family history of “lung disease”, and usually sleeps in a sitting position with the aid of pillows.
Current tests

The following tests are conducted: Chest X-ray (CXR), full blood count (FBC), urea, electrolytes and creatinine (UECs), arterial blood gases (ABGs) taken on room air, electrocardiogram (ECG) and pulmonary function tests (PFTs). A sample of sputum is collected for culture and sensitivity.

The CXR shows a flat, low diaphragm, bullae and some consolidation of the right middle lobe. The PFT results are: FEV₁ 1600 cc (predicted 3300 cc), and FVC 3400 cc (predicted 4100 cc). The patient also has an increased total lung volume and decreased tidal volumes. The initial sputum sample results are ++ gram-positive cocci.

The results of the blood tests are shown below:

<table>
<thead>
<tr>
<th>Normal values</th>
<th>Patient’s values</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH (7.35-7.45)</td>
<td>7.29</td>
</tr>
<tr>
<td>PaCO₂ (35-45 mmHg)</td>
<td>75</td>
</tr>
<tr>
<td>HCO₃⁻ (21-28 mmHg)</td>
<td>36</td>
</tr>
<tr>
<td>PaO₂ (80-100 mmHg)</td>
<td>49</td>
</tr>
<tr>
<td>O₂ Saturation (95-100%)</td>
<td>78%</td>
</tr>
<tr>
<td>RBC count (3.8-5.5 million/mm³)</td>
<td>6.8</td>
</tr>
<tr>
<td>Hb (14-18 g/dL)</td>
<td>19</td>
</tr>
<tr>
<td>Hct (42-52%)</td>
<td>57%</td>
</tr>
<tr>
<td>WBC count (5,000-10,000/mm³)</td>
<td>14,000</td>
</tr>
<tr>
<td>Chloride (98-106 mEq/L)</td>
<td>98</td>
</tr>
<tr>
<td>Sodium (136-145 mEq/L)</td>
<td>138</td>
</tr>
<tr>
<td>Potassium (3.5-5.3 mEq/L)</td>
<td>4.0</td>
</tr>
<tr>
<td>Urea (10-20 mg/dL)</td>
<td>19</td>
</tr>
<tr>
<td>Creatinine (0.6-1.2 mg/dL)</td>
<td>1.0</td>
</tr>
</tbody>
</table>

1. Discuss the defence mechanisms present in the respiratory tract to minimise or prevent retention of particulate matter. Which of these defences are damaged in smokers and why? How does this contribute to the development of COPD? Does Mick have any other risk factors?

2. Explain the results of the PFTs.
3. Explain the patient’s FBC results.

4. Determine the patient’s acid-base status and provide an explanation for the ABG results. Is Mick in respiratory failure? If so, why?

5. Explain the patient’s signs and symptoms based on your understanding of the pathophysiology of pulmonary emphysema. Which of Mick’s clinical signs and symptoms indicate an obstructive disease?
6. How do patients with chronic bronchitis differ from patients with emphysema in terms of their presentation and pathophysiology? Explain the ‘blue bloater’ presentation.

7. Discuss the management of COPD generally, and this patient in particular.